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ABSTRACT

This experiment investigated the capability for movement and muscle spindle function at successive stages during the development of ischemic nerve block (INB) by pressure cuff. Two male subjects were observed under six randomly ordered conditions. The duration of index finger oscillation to exhaustion, paced at 1.2Hz., was observed on separate days immediately, 5, 10, and 15 minutes following cuff application, and after kinesthetic sense loss (KSL). A control condition observed only reflex responses to sudden flexions and extensions of the relaxed finger at the above times during a single cuff application with no active movement. These reflex responses were also observed before cuff application and following movement on other days. Finger movement persisted for two or more minutes at each interval before KSL, and just under one minute following KSL. Reflexes appeared uninfluenced by cuff application without movement. Following movement during cuff application, reflexes were not found beyond the ten-minute observation. These findings indicate reduced but experimentally useful movement capability following KSL provided the subject rests. The alteration of muscle spindle function following movement before KSL is relevant to studies using INB to study the role of proprioception in skilled movement. (Author)

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Ischemic Nerve Block

The question of the role of proprioception in the performance of human movement is raised in virtually every course or conference on motor learning. The direct test of movement in the absence of proprioception has been conducted on experimentally deafferented on monkeys (Taub, Psrella, and Barro, 1973) and has been observed on deafferented man since the experiments of Lashley in 1917. Experimentation on man has been conducted with extreme difficulty since deafferentations have had to be either adventitious due to peculiar injuries, restricted due to specific localization of effects as in joint-replacement or anesthetization, or even questionable in completeness as in ischemic nerve blockage. Adding to these difficulties is the problem of clearly avoiding impairment of the efferent pathways, either directly in the application of regional nerve blocks or indirectly by the impairment of the muscle spindle system in its dual role as a specialized sense organ with involvement in determining (at least in part) muscular action.

There are two primary levels at which inquiry into the role of proprioception might proceed. The long-standing series of experiments on performance capabilities of variously deafferented humans may well continue. That research, forced as it is to primarily rely upon reversible deafferentation techniques, must drop back momentarily to a technological level to ascertain more surely exactly how each deafferentation

technique functions. This is so because there are numerous methods in use, each with its own constellation of specific effects and side effects. These techniques include blocking of distal joints by xylocaine ring block (Browne, Lee, and Ring, 1954) and ischemic nerve block at the wrist (Goodwin, McCloskey and Matthews, 1972), cooling of musculature (Paillard and Brouchon, 1974), differential axillary blockage of small diameter fibers (Smith, 1969) and a contentions ischemic blockage applied at the upper arm extensively used by Laszlo (1966). The comparability of these techniques has only recently been studied (Williams, 1977) in the search for the most effective and acceptable method of reversibly eliminating proprioception.

At yet another level of application of deafferentation, there is concern to demonstrate the compelling experience of attempting movements while reversibly deafferented. This demonstration brings to life the lines used to represent feedback on the ubiquitous human performance models we and our students view. Only then can the reality of deafferentation faced by accident victims, users of prostheses, workers in frigid environments, etc., be appreciated. The ischemic nerve block is widely used at both demonstrative and research levels, and the present paper relates to a long-standing controversy regarding that technique. The primary controversy over the ischemic nerve block lies in the assumption (Laszlo, 1966) that sensory elimination precedes significant reduction in motor capability. Attempts to resolve that issue by experimentation

(Kelso et al., 1975) and by debate (Bairstow and Laszlo, 1976; Kelso, Stelmach and Wannamaker, 1976) have proceded without evidence on one key issue, namely, the effect on performance of systematic change in the amount of neuromuscular activity during the development of ischemia. The issue appears to underlie the contradictory findings of tapping capability found in Laszlo's subjects but to a lesser extent in the Kelso et al. subjects. Laszlo's procedure stipulated rest during the cuff application while Kelso systematically applied supra-maximal shocks in some experiments or cumulatively assessed tapping performance in another experiment. The likely accumulation of fatigue within two minutes of performing light tasks such as handwriting or piano playing under the pressure cuff had been observed by Merton (1956). Merton found that two minute limit of movement immediately after occlusion. It is important to establish whether there is less movement capability if activity is commenced even later in the occluded period, especially after the loss of kinesthetic sensation. Better knowledge of the amount of activity possible after kinesthetic sense loss and the amount of activity allowable during the development of ischemia will allow the design of more appropriate experiments and demonstrations using the ischemic nerve block.

The present experiment therefore systematically observed index finger tapping to exhaustion by allowing total rest prior to tapping at the time of cuff application and at longer durations of ischemia up to the time of kinesthetic sense loss.

Secondarily, the state of the muscle spindle loop was observed by stretch reflex elicitation before cuff application and following tapping. This also served to verify this aspect of kinesthetic sense loss.

Method

Subjects and design. The author and an assistant served both as subjects and experimenters. Both were experienced with the pressure cuff. Five index finger stretches to sudden flexion and extension were applied prior to cuff application and following tapping. On five occasions separated by seven-day intervals, tapping observations were made, in randomized order, either immediately, 5, 10, or 15 minutes after cuff application or at the time of kinesthetic sense loss. Duration of tapping at 1.25 Hz was the primary dependent variable. A single control session observed reflexes at each of the above times during ischemia in the absence of tapping.

Apparatus. The index finger was fixed in an aluminum groove, open underneath, and pivoted horizontally above the metacarpophalangeal joint to allow a "tapping" (flexion and extension) motion. The remainder of the hand was fixed, fingers straight and thumb uppermost to isolate movement at the metacarpophalangeal joint. The finger splint axle turned a potentiometer which signalled angular motion during tapping as well as the onset of movement in reflex elicitation. A metal pointer extended forward from the finger splint for 25 cm such that the arc of its tip was adjacent to a vertical

screen. Two diode lights, 30° apart on the screen and lit alternately at 300 msec intervals served to pace the tapping movements.

Reflexes were elicited by means of a spring-generated extension or flexion of the finger. The spring was held taut by an electromagnet and released instantly when current was broken. The spring could be attached via pulley and cord to either side of the finger splint, thereby providing sudden movements of flexion or extension. Bipolar surface electrodes (Litton silver chloride) were positioned above the motor points of flexor digitorum longus and extensor digitorum to record stretch-elicited muscle action potentials.

Analog records of muscle action potentials and displacement were simultaneously recorded on a Litton (218057) pen recorder and on magnetic tape (Hewlett Packard 3960). Reflex quantification was made from tape playback on a storage oscilloscope (HP-184A) while duration of movement was based on the pen record.

Procedure. The ischemic nerve block was produced from a sphygmomanometer cuff applied to the upper arm and inflated to 180 mm Hg following the procedure detailed by Laszlo and Bairstow (1971). Testing for kinesthetic sense loss was restricted to the index finger tip and the near side of the hand proximal to the metacarpophalangeal joint since the remainder of the hand was secured in the apparatus three or four minutes prior to the experimental observation of the day. Tactile sensation was tested by light touch using the

experimenter's finger. Awareness of passive movement was tested by moving the relaxed finger at varying speeds while in the splint. When active movements were inaccurately perceived, kinesthetic sensation was declared to be absent and assessment of movement capability began. The alternating pacing lights were initiated and visually-guided tapping commenced. As fatigue developed, tapping rate was primarily maintained but movement amplitudes, especially to extension, were permitted to diminish. Following tapping, stretches were applied alternately to flexion and extension to elicit 5 reflexes from extensor and flexor muscles respectively. Due to mild discomfort, circulation was restored following the post-tapping reflex observations.

Results

Tapping. The duration of tapping, based on the loose criterion of eventual inability to move, is shown in Table 1. The durations shown include movements which were becoming smaller in amplitude as well as slowed in rate. Even so, the indications are that movement capability declines from approximately 3.5 minutes when commenced immediately after cuff application to about 2 minutes after 15 minutes of occlusion. By the time of kinesthetic sense loss at about 22 minutes, the duration of tapping was further reduced to less than one minute. Similar trends are apparent for both subjects.

Insert Table 1 about here

Details of tapping performance are displayed in Figure 1. Successive records illustrate the amplitude of tapping movements and show the tapping capability diminishing to virtual paralysis. Both duration and amplitude of tapping appear considerably reduced following kinesthetic sense loss, especially for finger extension. There was no recovery of movement following each tapping attempt until circulation was restored. In summary, a gradual decline in duration of tapping capability was found as duration of occlusion increased, and an even shorter duration of notably impaired movement was found after kinesthetic sense loss.

Insert Figure 1 about here

Reflexes. There was relatively stable persistence of stretch reflexes in the control session without movement with the exception of the disappearance of this reflex in the extensors following kinesthetic sense loss. This state is indicated in Table 1 by the mean reflex latencies found at each duration of occlusion. The effect of movement prior to reflex assessment at varying lengths of occlusion was to cause the earlier disappearance of the reflex response. Even after 10 minutes of occlusion, it was not possible to elicit reflex responses following tapping to exhaustion. It is not possible to know where in the reflex loop this impairment arose using the present procedure. Nor was it possible to discern whether tapping altered other sense receptors or the

time of kinesthetic sense loss because circulation was restored following post-tapping reflex observations.

Discussion

The finding of severely limited duration and amplitude of tapping even after complete rest during occlusion clarifies the claims made by previous researchers. Provided minimal extensor activity is required, about 50 taps appear possible following kinesthetic sense loss. This performance is similar to the tapping performance with minimal training reported by Laszlo and Manning (1970) and also to the performances reported by Kelso et al. (1974) in the 50% of their subjects who were able to tap.

The Kelso et al. (1975) claim of diminishing motor capability concurrent with sensory reduction is also supported: using repeated 10-tap tests at 0, 5, 10, 15, and thereafter one-minute intervals, they indicated a sharp decline in the number of taps at about 22 minutes by which time approximately 100 taps would have been completed. The present results confirm the limited tapping performance after a similar length of occlusion, even when subjects rested during the occlusion. Merton's (1956) observation of a finite movement capability soon after occlusion is also extended to show gradually reducing capability as the occluded period extends.

The ischemic nerve block is thus indicated to have limited experimental potential on the grounds of impaired movement capability at the time of kinesthetic sense loss even

following complete rest. More specific blockage of the spindle system by dilute xylocaine in conjunction with interruption of specific sense receptors at the joint would appear to provide more certain deafferentation in conjunction with more normal movement capability (Williams, 1977).

As a demonstration technique, the ischemic nerve block might be expected to provide approximately 30 seconds of simple activity requiring little finger extension. In the author's experience, and as reported by Laszlo and Manning (1970) and Kelso et al. (1974), about 50% of subjects will either show significantly poorer performance (Laszlo's Beta subjects) or incapability of tapping.

A further variable which may maximize movement capability following sensory reduction by ischemia is cuff pressure. Gelfan and Tarlov (1956) reported that ischemia selectively blocked the small diameter fibers earliest while nerve compression first affected large diameter fibers (including alpha efferents). Thus, systematic variation of cuff pressure for each subject might reveal a pressure different from the recommended 180 mm which would be sufficient to create ischemia while minimizing compression.

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Table 1

Duration of Tapping (min:sec) While Occluded

	Commencement of Tapping				
	Immed.	5 min	10 min	15 min	Kinesthetic Sense Loss
Subject 1	3:40	3:10	3:15	2:45	0:42
Subject 2	3:20	2:30	2:55	1:58	0:53

Table 2

Mean Latencies (msec) for Stretch Reflex

Following Tapping to Exhaustion

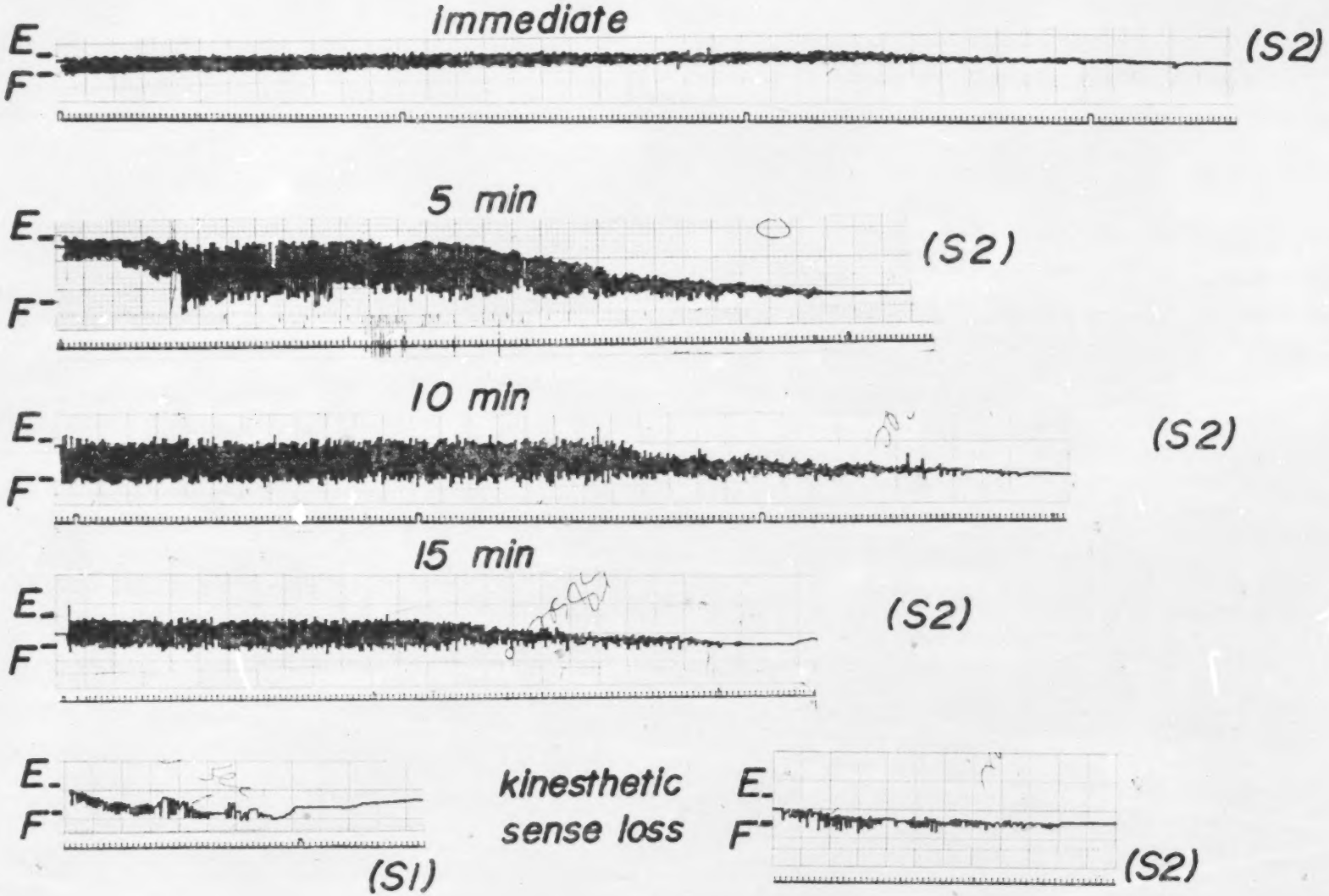
	Time of Elicitation					
	Pre-cuff	Immed.	5 min	10 min	15 min	Kinesthetic Sense Loss
Single Control Session Without Tapping						
Flexors	33.3*	30.0	29.15	28.3	30.8	30.0
Extensors	43.23*	45.8	47.15	51.6	38.6	abs**
Following Tapping to Exhaustion						
Flexors	33.3*	33.0	54.5	abs	abs	abs
Extensors	43.23*	73.0	52.5	abs	abs	abs

*Pre-occlusion means were obtained from averaging all pre-cuff observations.

**abs denotes no reflex found

Figure Caption

Figure 1. Sample tapping records after each duration of occlusion.


Immediate
E
F
(S2)
5 min
E
F
(S2)
10 min
E
F
(S2)
15 min
E
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(S2)
E
F
(S1)
kinesthetic
sense loss
E
F
(S2)